for pleurisy, showed that the heart presented no anomaly before the accident. Mendelsohn thinks that in addition to the traumatism there was a strain on the heart from the struggle, and that this, with the anxiety of the man's critical position, was important in the production of the lesion.

Tuberculous Endocarditis.—BENDA (*Deutsche med. Wochenschrift*, 1898, No. 7) records a remarkable example of this. A boy, aged ten years, died of acute miliary tuberculosis following hip disease. The numerous recent tubercles in all organs pointed to a blood-infection, but examination of the veins showed no point of entrance. There was a warty endocarditis, and on the mitral valve, besides the fine translucent granulations, there was a rough yellow nodule the size of a hempseed. This was composed of a vascularized granulation-tissue with epithelial cells, but with no giant cells. The free surface showed irregular necrotic areas, partly caseous and partly hyaline, with enormous numbers of tubercle bacilli. A zone half a millimetre in thickness consisted wholly of colonies. That this was the source of the general infection cannot be doubted.

Dental Caries and Soil.—The intimate relation of dental caries with other important diseases makes interesting a reference to certain observations of C. ROSE, which have recently been confirmed in Sweden. A year ago Rose claimed, as the result of the examination of the teeth of 20,000 Bavarian recruits, that the harder the water and richer the soil in lime and magnesia, the better the teeth. Forberg reported the Swedish results at a recent congress of dentists. Examinations of the teeth were made by various dentists in different parts of Sweden. In that country the racial and economic conditions are tolerably uniform, but the hardness of the water varies greatly. A constant increase of the proportion of carious teeth was found to coincide with the diminishing hardness of the water, so that with a hardness of 12° there were 15.4 per cent.; with a hardness of 0.5°, 25 per cent. of carious teeth.—*Münchener med. Wochenschrift*, 1898, No. 3.

Typhoid Fever without Intestinal Lesions.—NICHOLS and KEENAN (*Montreal Medical Journal*, January, 1898, p. 9) report the following case of typhoid fever without intestinal lesions: The patient came to the out-door department, stating that for about two months he had been suffering from headache, loss of appetite, and general weakness. Had been forced to give up work several times.

On admission his temperature was 103.6°, pulse 104, and respiration 36. Tongue was coated and dry; abdomen distended, tense, and tender. Fading rose-spots were visible, and the spleen was palpable. Bowels constipated. There was some impairment of the percussion note in left axillary region. The blood gave the typical serum reaction. The patient gradually failed, and death occurred thirteen days after admission. Blood-cultures taken before death were negative.

The chief points in the autopsy report are as follows: Spleen was dark-red and pulpy. Cultures from the spleen gave a bacillus which fulfilled all the requirements of Eberth's bacillus. The intestines were entirely free from ulceration, although the lowest three Peyer's patches in the ileum were slightly raised, but showed no signs of ulceration. There was no evidence of healed typhoid ulcers. Mesenteric glands were generally enlarged, congested, and succulent, especially about the ileo-cæcal region. Left lung showed broncho-pneumonia. Cultures from the liver were positive for Eberth's bacillus. Blood cultures taken at the autopsy were negative.

A search of the literature for the past ten years showed only nine cases of typhoid fever without intestinal lesions, which had been confirmed by the discovery of Eberth's bacillus. A number of other cases are reported, which, however, are not included, owing to the lack of corroboration by bacteriological investigation.

Typhoid without intestinal lesions falls clinically into three main classes:

1. Typical typhoid, minus the ulcerations.
2. Spleno-typhoid.
3. The nervous type, with extreme intoxication.

The cases of the first type are rare. Diarrhœa may be present. Three of the nine cases belong to this group.

The second type was first described by Eiselt. It is characterized by an excessively large spleen, often with acute perisplenitis and fever of a recurrent type. The writers state that some of these cases do present ulceration of the intestines, but it is often absent. Two of the nine cases belong to this class.

The third class, due to severe intoxication, are characterized by extreme prostration, delirium, coma, sometimes hyperpyrexia, degenerative changes in the vascular system leading to purpura, hæmaturia, melena. Jaundice is sometimes present. Many of these cases are, no doubt, examples of secondary septic infection. This class includes the rest of the nine cases, including the one the writers report.

Nichols and Keenan discuss at considerable length the possible explanations for the absence of intestinal lesions in typhoid. Three hypotheses must be considered. First, one must think of the possibility of infection through some avenue other than the intestine, as, for example, the respiratory tract. In such cases the brunt of the disease might fall on the lungs, and the intestine escape. Secondly, certain ptomaïnes derived from the gastro-intestinal tract, either circulating in the blood or present in the intestinal mucosa, act so as to neutralize the local action of the typhoid virus and bring about intestinal immunity. Finally, it may be assumed that toxins derived from germs other than typhoid may antagonize their virus, and a local immunity be thus acquired.

On the Coincident Occurrence of Epilepsy and Diabetes Mellitus.— Ebstein (*Deutsche medicinische Wochenschrift*, 1898, No. 1, S. 3, u. No. 2, S. 22) published a communication bearing on this subject in the *Semaine Médicale*, May 6, 1896, based on a case which came under his personal observation. Since then four other cases have been observed, the notes of which are here recorded. Three possibilities present themselves in considering the association between epilepsy and diabetes mellitus. First, the epileptiform attacks may be caused by some toxic substance occurring in diabetes; second, the diabetes mellitus or glycosuria may be a result of the epileptiform attacks; and, thirdly, both epilepsy and diabetes mellitus may be dependent upon some common etiological factor.

Considering the first possibility, Ebstein states that it is not unreasonable to suppose that convulsions, due to the toxic influence of the products of metabolism, might be expected to occur in diabetes mellitus, just as convulsions occur in the uræmia of nephritis. Personally, he has never observed such instances. Reference is made to a case recorded by Finlayson, in which repeated convulsions occurred in a case of diabetic coma seventeen hours before death. Dreschfeld observed convulsions once in sixteen cases of diabetic coma, and in eighty cases of this complication of diabetes collected from the literature convulsions occurred in six instances. G. W. Jacoby considers the epilepsy in diabetics to be an intoxication epilepsy due to the circulation of acetone in the blood, and terms the condition epilepsia diabetica.

Those cases of diabetes mellitus which may be said to have been occasioned by the epilepsy may be divided into two classes: first, cases in which temporary glycosuria occurs after the epileptiform seizure; and secondly, those in which a more or less permanent glycosuria or diabetes mellitus ensues. Ebstein has never seen either a temporary or permanent glycosuria which could be attributed to the epileptiform seizures, and Griesinger's experience agrees with his own.

The third possibility, namely, that the diabetes and epilepsy may be dependent on some common cause, and not one on the other, is then discussed. The causes in these cases may be general or local. The most important of the general influences is heredity and a family predisposition, which plays so great a part in the etiology of both diabetes and epilepsy. Ebstein's first observation illustrates the effect of heredity. The patient's mother had softening of the brain and a brother epilepsy, though no diabetic history is stated to have been obtained. The patient had both diabetes and epilepsy, the latter appearing much later than the former, though, as Ebstein thinks, not dependent upon it. Ebstein states that although cases of diabetes and epilepsy are not uncommon in members of families with neurotic histories, yet it is a point worthy of note that these two diseases rarely make their appearance simultaneously.

The second and third of Ebstein's observations illustrate those cases where the epilepsy and diabetes result from a common local cause, in these particular instances a localized brain lesion. Both were cases of Jacksonian epilepsy, associated with diabetes mellitus decipiens intermittens. The amount of sugar was in both instances very small, and appeared intermittently. In Case 2 the largest quantity of sugar excreted in the twenty-four hours was only 8.8 grammes, while in Case 3, 14.5 grammes was the largest quantity excreted. His fourth case was one in which the epileptic symptoms began apparently as a petit mal, developing later into a grand mal. In this case also the diabetic symptoms were intermittent in character. The amount of sugar is not stated.

The urine examinations in Cases 2 and 3 were made with the greatest care. Daily analyses extending over months are recorded. Ebstein states that in these cases greater care in performing the sugar-tests is required than is usually observed in examining for sugar. Not only must daily examinations of the urine be made, but each amount of urine voided should be analyzed, otherwise sugar is likely to be overlooked in the urine. From his personal experience Ebstein is led to believe that glycosuria or diabetes mellitus decipiens intermittens will be found more frequently in epilepsy, especially the Jacksonian type, than heretofore, if due care in making the urinary examinations is exercised.

Spontaneous Rupture of a Fatty Heart.—GREIG (*The Canadian Practitioner*, February, 1898, p. 81) records a case of rupture of the heart occurring during the act of passing a soft-rubber stomach tube for purposes of lavage in the treatment of a gastritis from which the patient was suffering. The patient was a woman, sixty years of age, and not particularly stout. The tube had just been passed into the stomach, without any excessive retching or straining on the part of the patient, when suddenly a pallor spread over the patient's face and her eyes rolled up. Realizing that something had happened the tube was immediately removed, the patient was placed on her back, and every possible means used to resuscitate her, but she was dead. At autopsy the heart was found ruptured, but unfortunately it is not stated into which of the cavities of the heart the rupture occurred. Extensive fatty changes of the heart and liver were found.

Greig states that the case is a unique one, and so far as he could ascertain no similar case had ever been reported.

The Use of Sudan III. as a Staining in Clinical Microscopy.—RIEDER (*Deutsche Archiv für klinische Medicin*, Band lix., 3 u. 4 Heft, p. 445) highly recommends Sudan III. as a means of demonstrating fat, occurring normally in the tissues or as a product of tissue or cell degeneration. It is a diazzo-coloring material, and was first prepared by Nietzki in 1880. L. Daddi, of Turin, in 1896 first pointed out the value of Sudan III. as a means of demonstrating fat, which it stains a beautiful scarlet-red color. It is a light, reddish-brown powder, which is insoluble in water, but easily soluble in alcohol, ether, chloroform, xylol, fatty and ethereal oils. When fat is once stained with the dye, it retains it with great tenacity and is decolorized with only the greatest difficulty.

The technique of using the stain in demonstrating fat in histological and pathological sections is first dealt with. Rieder lays special stress on the advantage of using Sudan III. in staining fat particles in human secretions and excretions. Large fat droplets take a bright-red, and small droplets only a yellow or orange-red color. Strong solutions used for some time stain the fat a dark or scarlet-red, whereas weaker solutions yield a light yellow color.

In clinical work the stain is very serviceable in demonstrating fat in cases of lipæmia, lipuria, and chyluria; also in the stomach-contents, in the feces of infants and of adults with icterus; in the sputum of patients suffering from bronchiectasis and lung abscess; and finally, and most important of all to the clinician, the fat in finely, coarsely granular and fatty casts and fatty renal epithelial cells in chronic parenchymatous degeneration of the kidneys.

Rieder recommends a concentrated solution of Sudan III. in 96 per cent.